A Contribution to the Study of Paroxysmal Hæmoglobinuria.—Donath (*Zeit. f. klin. Med.*, 1904, Bd. lii., Heft 1 and 2) reports very fully three cases of paroxysmal hæmoglobinuria induced naturally and experimentally by exposure to cold, and reviews the various attempts that have been made to explain the origin of this condition. In numerous experiments made with suspensions of red blood cells drawn from the finger before, during, and after attacks, he was unable to demonstrate any abnormal susceptibility of the corpuscles to cold, agreeing with the observations of Ehrlich, Boas, Chvostek, and others. Contrary to the findings of Lichtheim and Chiaruttini, there was no solution of the hæmoglobin when the blood was coagulated at various temperatures. All these cases showed slightly diminished resistance of the red blood corpuscles to mechanical insults, but the results varied considerably in the same case at different times, and there was a marked individual difference between the several cases. The variation from normal was always small and altogether out of proportion to the ease with which exposure to cold precipitated an attack. There was no such susceptibility to mechanical influences as apparently existed in the cases of Chvostek and Chiaruttini. Hæmoglobinuria occurred in one case after simple ligature of the finger or arm. In the other two cases the procedure gave negative results. Luzzatto and Sargente have shown that this hæmoglobinuria following stasis is not due to mechanical injury, but to thermic influences, the solution of the hæmoglobin not occurring when the normal temperature of the ligatured extremity is maintained. Donath was unable to demonstrate any direct decrease of resistance in the red blood corpuscles to cold or mechanical influences. He attempted to produce evidence in support of Ehrlich's view that in these cases under the influence of cold the blood serum gained a red blood corpuscle dissolving property. The serum obtained during an attack and during the interval was tested in varying dilutions with the red blood cells of the same individual, of normal individuals, and of patients with other diseases. In several instances the serum showed a slight hæmolytic effect on the cells of other individuals, but the effect was the same with the interval serum and the attack serum, and in no way above the normal isolytic action exhibited by various sera. In only one instance did the attack serum show a hæmolytic effect slightly above the interval serum.

In none of the three cases did the interval serum show autolytic properties, and although this is opposed to results published by other authors, he concludes that the property must at least be variable and cannot be accepted as the rule. In one case the attack serum showed a slight autolytic property.

Donath points out that although he has been unable to demonstrate the presence of a hæmolysin in his cases, this is no argument against the existence of such a property; for if a toxin be formed there is opportunity enough for it to become immediately anchored, and it could be demonstrated only if present considerably in excess of the amount bound by the susceptible red blood corpuscles, *i. e.*, those with suitable receptors. Attempts to "activate" inactive interval serum by the addition of attack serum, supposing complement to be produced in excess during an attack, proved fruitless, as did also efforts to reactivate inactive attack serum by adding active interval serum or active normal serum on the supposition that amboceptors might be produced in excess.

In order to see if the red blood corpuscles themselves were more susceptible to hæmolytic influences, he subjected the cells from his cases to serum from other individuals, to hæmolytic immune serum and to certain red blood corpuscle dissolving substances, such as abrin and ricin. The results show that the red blood cells in his cases have the same relation to human serum from other sources and to hæmolytic immune serum as the erythrocytes of other cases, while abrin and ricin agglutinate them in somewhat weaker solutions. By collecting serum from the arm of different patients after having put on a compression ligature and immersed them in cold water for from six to ten minutes, he was able to show in two out of four cases a slightly increased hæmolytic action of such serum on the red blood corpuscles, not only of the hæmoglobinuria cases, but also of a number of other patients.

Donath thinks he is justified in concluding that the blood disintegration does not follow as a result of the direct action of cold or of purely mechanical influences upon the red blood corpuscles, but is due to hæmolytic properties of the serum. He believes this property to depend upon substances similar to the serum hæmolysins.

Pneumococcus Peritonitis.—ANTON GHON (*Wiener klin. Woch.*, 1904, xvii. p. 267) reports four cases of acute fibrinopurulent peritonitis due to diplococcus pneumoniæ. The first case was that of a girl, aged twenty-three years, who was taken suddenly ill with pain in the abdomen and died on the fourth day of general peritonitis. Autopsy showed a diffuse fibrinopurulent peritonitis and a perforated gastric ulcer. Cultures from the exudate gave a pure growth of pneumococcus. In Case II. there was a diffuse serofibrinopurulent peritonitis following perforation of a gastric carcinoma. Cover-slips from the exudate showed very numerous definitely lancet-shaped diplococci, retaining the Gram stain, and a small number of bacilli. The colonies on agar plates were chiefly of pneumococcus. Case III. was one of diffuse fibrinopurulent peritonitis developing eight days after gastroenterostomy for carcinomatous obstruction of the pylorus. Cover-slips from the exudate showed principally Gram-positive lancet-shaped diplococci and a few short bacilli. In cultures only a bacillus identified as colon grew, but inoculation of the exudate into a mouse produced death in twenty-four hours from pure pneumococcus septicæmia.

In Case IV. there was a deeply ulcerated gastric carcinoma without perforation, marked secondary hepatic and glandular involvement, fibrinopurulent peritonitis localized about the region of the pylorus, and fibrinopurulent meningitis. Cultures from the meninges and the swollen glands along the greater curvature gave a pure growth of pneumococcus. Cover-slips from the peritoneal exudate showed only lancet-shaped Gram-positive diplococci, but in cultures there were also a few colonies of colon bacillus and two colonies of staphylococcus aureus.

Although in three of these cases the pneumococcus was not found in pure culture, Ghon feels justified in assuming that it was the active agent. He quotes Jensen, who, in twenty cases of pneumococcus peritonitis, found three following perforation of gastric ulcer, and Brunner, who collected twenty-two cases of perforated gastric ulcer, examined bacteriologically and found four due to pneumococcus. Ghon considers the mouth and pharynx the source of infection. In Case III. there